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## CASE REPORT: Arterial Gas Embolism Induced Ageusia

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This article details a previously unreported arterial gas embolism (AGE) clinical presentation. A military aircrew candidate water survival course trainee lost his sense of taste ("ageusia") after only a few compressed air breaths and ascending from the shallow depth of four feet. Ageusia was his only AGE sign or symptom.

Rapidly diagnosing and treating AGE and other decompression illnesses is essential for positive patient outcomes and requires understanding the anatomic and physiologic effects of atmospheric pressure changes.

**Keywords:** decompression sickness (DCS), decompression illness (DCI), arterial gas embolism (AGE), barotrauma, ageusia, anosmia, hyperbaric oxygen treatment (HBO), US Navy Treatment Table 6 (TT6), aircrew candidate (ACC).

### Introduction

Physicians must react quickly to decompression illnesses (DCI), comprised of decompression sickness (DCS), arterial gas emboli (AGE) and dysbaric injuries, because they can be rapidly fatal. Military physicians may be DCI trained if they routinely care for flying, diving or other special duty personnel; however, most civilian primary care and emergency physicians lack DCI training. Unfortunately, civilian recreational divers are 96 percent more likely to develop DCS and AGE than military divers (4). This disparity may be due to enhanced military diver training, supervision, equipment, or medical and physical statuses relative to civilian divers, but anyone who dives deeper, more frequently, or surfaces faster than recommended can develop DCI. This case highlights the need to include DCI in differential diagnoses if patients present with any signs or symptoms after inhaling compressed gas, regardless of symptom severity or water depth. This case report describes the event history and clinical course of a military trainee who developed an AGE after taking just a few compressed air breaths in only four feet of water. The case's

history and symptoms were unusual, previously unreported in the literature, and accentuate the need for physicians' DCI awareness.

### Case Report

A 20 year old black male, helicopter aircrew candidate (ACC) was referred by a dive master to the flight surgeon's office at Fairchild AFB, Washington for an unusual complaint, loss of the sense of taste ("ageusia"). The patient was egressing a mock helicopter fuselage "dunker" submerged four feet in a pool. Soon after surfacing the ACC ate a fruit-filled pastry while waiting for his next training event. Unable to taste the food, the ACC notified the dive master; he called the clinic and was directed to immediately escort the ACC to the flight surgeon (FS) for evaluation.

The ACC recounted to the FS that he had been in the dunker at four feet depth and could not taste food a few minutes after surfacing; he offered no other information. With specific questioning the ACC noted a diminished sense of smell ("anosmia"), and that immediately after surfacing he felt "strange", and gasses "moving in his head". With additional definitive questions the ACC divulged that his egress had been slow and he required several emergency breaths of compressed air prior to surfacing. A final question resulted in the ACC admitting he held his breath while surfacing. He denied any other symptoms including pain, dizziness, nausea, shortness of breath, abnormal skin sensations, or arthralgias. He was a one-pack per week smoker.

Physical exam vital signs were normal. He was alert, oriented, and answered questions appropriately. Mood was euthymic. Tympanic membranes were bilaterally sluggish during valsalva. Sinuses were non-tender to percussion and there were no signs of head or neck trauma. Respiratory, cardiac, and orthopedic examinations were unremarkable. Neurological exam was remarkable for his stated inability to taste sugar or salt grains placed on



his tongue, despite detecting their gritty texture. Sense of smell was not definitively evaluated during this exam. The remainder of the exam was normal. Chest radiograph was normal and had no pneumothorax signs.

The initial differential diagnosis included sinus block, AGE, cerebrovascular accident, TIA, conversion disorder, anxiety reaction, malingering, compressed air and/or pool chemical induced dysgeusia, and hypoxic anomaly. The Brooks AFB, Texas hyperbarics fellow and attending physician were contacted; they recommended the FS assume the ACC had an AGE and immediate treatment with U.S. Navy Treatment Table Six (TT6) hyperbaric oxygen (HBO). The nearest HBO facility was in the Spokane, Washington Deaconess Medical Center. The Deaconess hyperbaricist was told of the case and Brooks AFB guidance, and agreed with the AGE diagnosis and HBO treatment. The patient was given 100% oxygen by aviator's mask and transferred immediately.

Upon arrival at Deaconess the ACC underwent TT6 HBO. Apple and grape juice samples were placed in the chamber with the patient. At sixty-foot sea water (2.8 atmospheres) he sipped the juices and easily recognized both flavors after five minutes at 2.8 atmospheres. The patient completed TT6 and was told to see his FS the next morning. The next day the patient's baseline taste and smell senses remained intact. He was advised not to fly or travel to higher altitudes for 72 hours, and given appointments for studies recommended by Brooks AFB physicians (head and chest CTs, echocardiogram, neurology and otorhinolaryngology consults).

The chest and head CTs revealed no stroke, sinusitis, pneumothorax or other lesion indications. An agitated saline echocardiogram found no patent foramen ovale (PFO), atrial septal defect or other defect that could predispose a right to left shunt.

Several days after HBO the neurologist identified no permanent sequelae, or cranial nerve or cortex defects. He suggested the patient's ageusia may have been secondary to anosmia, but concurred that an AGE was the proximate cause. The otorhinolaryngologist found no sinus infections or other pathology, and also concurred with the "decompression syndrome" diagnosis. Both consultants recommended the ACC return to helicopter training after FS clearance. Follow-up two months after the DCI episode indicated no symptom recurrence, and

the ACC continued his flight crew training without incident.

### Discussion

There are over one million sport divers in the United States and fifteen million world-wide who perform about 250,000,000 dives annually (4). Currently in the US there are approximately 1000 cases of DCI per year with a ten percent mortality rate (9). The US sport diving population annual risk of death is 1/6250 divers and the annual risk of developing DCI is 1/2900 divers (4). DCI odds are much lower for highly trained and supervised military divers whose risk is 1/76,000 divers per year (4). The patient in this case report had no SCUBA experience before his underwater egress training.

DCI is a broad category of illnesses and injuries that can occur when divers, fliers, climbers or travelers encounter reduced atmospheric pressures (3). DCI includes dysbaric injuries, Type I and Type II DCS, and AGE (12). The primary physiologic results of reduced atmospheric pressure are the formation and expansion of nitrogen bubbles. Normally nitrogen is dissolved in body fluids and tissues, but decreased atmospheric pressures allow nitrogen bubbles to evolve and precipitate DCIs (13).

Dysbaric injuries are barotraumas due to pressure effects on various organs. Sinus and ear blocks, and tooth cavity pain are often experienced by descending SCUBA divers. Pulmonary barotraumas are also reported in the literature; one case report described delayed onset pulmonary barotrauma in a diver which resolved using TT6 HBO (7).

DCS signs and symptoms are categorized as Type I or Type II. Type I DCS includes "bends" or joint pain in 70-90 percent of cases; skin pruritic rashes (cutis marmorata) and lymphatic obstruction with skin pitting edema are less common (13). Type II DCS is more severe and can be rapidly fatal; it includes shock, pulmonary "chokes", and neurologic sequelae resembling cerebrovascular accidents due to nitrogen bubbles occluding blood supply to the brain or spinal cord.

AGEs cause serious injuries and are the second most common cause of sport diver fatalities (13). AGEs occur when gas bubbles form in the arterial or venous circulation travel to tissue end points and are commonly seen in SCUBA divers breathing compressed air, especially

those who hold their breath while ascending or whose ascent rate exceeds the ascent rate of their exhaled bubbles. Inhaled compressed air volume increases as divers ascend which increases lung tissue pressures and may cause pneumothoraces. Air may evolve into the mediastinum (mediastinal emphysema) or the skin (subcutaneous emphysema). Lastly, air bubbles may directly enter arterial vessels, flow into the cranial circulation, and impede blood flow to the brain or spinal cord (12). This process probably best describes the AGE etiology in this case report.

Anatomic variants, behaviors, injuries and illnesses increase AGE likelihood. Ten to twenty-seven percent of people have a patent foramen ovale (PFO) predisposing right to left shunting, bubbles entering the arterial circulation, and AGEs in divers (1, 3 and 5). Smoke and/or tobacco damaged lungs, COPD, emphysema, asthma, or prior pneumothoraces increase AGE probability (3 and 8). Smoking may have increased the ACC's AGE susceptibility.

The best current treatment for AGE and Type II DCS is immediate TT6 HBO starting with 100% oxygen. The patient is placed in a hyperbaric chamber and rapidly descended to 60 feet salt water (FSW) (2.8 atmospheres) which reduces the size and number of air or nitrogen bubbles, thereby reducing signs and symptoms. Gradually the pressure is decreased from 60 FSW (2.8 atmospheres) to 30 FSW (2.0 atmospheres) to ambient pressure (1.0 atmosphere), while alternating between 100% oxygen and room air to avoid oxygen toxicity (14). The Navy TT6 lasts approximately five hours and is absolutely necessary to treat AGEs (2 and 14). Despite rapid and proper diagnoses and treatment, mortality rates may still approach 50% (4).

Cranial nerve anatomy was important in this case. Portions of cranial nerves VII (facial, specifically chorda tympani), IX (glossopharyngeal) and I (olfactory) are involved in the sense of taste. This case's physicians surmised the ACC's cranial nerves were affected by an AGE. Compressed air induced internal carotid air bubbles were proposed as the AGE's proximate cause (15). The nuclei of cranial nerves VII from the pons and IX from the medulla may have been compromised via bubble barotrauma or blood flow occlusion. Another etiology could have been a gas bubble insult to the chorda tympani branch of cranial nerve VII. Spontaneous bubble formation in the cranial nerves or nerve nuclei was unlikely because

bilateral, simultaneous bubble barotraumas would have to occur to evoke total ageusia. Type II DCS (nitrogen bubble induced injuries) were also unlikely due to the depth and duration of the ACC's dunker training. Olfactory nerve or cortex insults, which was the neurology consultant's opinion, were plausible due to the senses of smell and taste being intimately related. Regardless of the actual location of the seminal lesion, the patient's primary symptom and chief complaint was ageusia. When treated with Navy TT6 HBO the ACC stated he rapidly regained his sense of taste and could distinguish apple and grape juice flavors.

Isolated cranial nerve findings in AGE are extremely rare. However, the literature includes a few examples. One patient developed an isolated cranial nerve III (oculomotor) palsy after SCUBA diving (10). Another diver developed optic nerve neuropathy affecting visual acuity and color vision (11). Patients undergoing cranial surgery, such as acoustic neuroma resections, have developed iatrogenic AGEs (6).

### Conclusion

While DCS and AGE are uncommon, if unrecognized and untreated, they can be rapidly fatal. Equipment problems, improper diving techniques, inexperience or panic may cause divers to hold their breath during ascent and induce AGEs (4). Pre-existing medical conditions may increase AGE susceptibility. Primary care, emergency and military doctors need to understand the etiologies and pathogenesis of DCIs. Understanding anatomy and neuroanatomy is critical to identify target organs compromised by DCI gas bubbles. Physicians should include DCI in differential diagnoses when patients have signs or symptoms after submersion to any depth or inhaling compressed air. Accurately relating physical exam findings and differential diagnoses to hyperbaric and other medical specialists is essential to obtain timely and proper DCI care. Treating physicians with minimal DCI experience can also access the Divers Alert Network online for rapid advice and recommendations (9). Immediate treatment is needed; the consequences of delayed treatment can be dire.

The patient in this case report may have had permanent ageusia/anosmia, or worse outcomes if treatment was delayed. Urgency to treat should override perfect DCI diagnostics. This concept was exemplified in this case by the less than scientifically rigorous attempts to evaluate anosmia, and blind the patient's encounters with salt, sugar and fruit juices during the physical exam and TT6. There were no significant reasons to suspect the validity of the pa-



tient's reported symptoms or responses to taste tests, and rapid assessment and treatment were critical for the patient's well-being.

Post-recompression evaluations are necessary to determine future atmospheric pressure change tolerances. This patient did well after TT6 HBO and no sequelae were seen. Special studies and consultants identified no lesions or persistent health deficits. The ACC was declared fit for flight and dive-related duties, and continued his training.

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